Role of Physiotherapy in Ventilator Induced Diaphragmatic Dysfunction: A Scoping Review

Lakshita Garg[1*], Nahid Khan[2], Noor Mohammad[2]
1* MPT Cardiopulmonary student, Department of Physiotherapy, SNSAH, Jamia Hamdard
2 Assistant Professor, Department of Physiotherapy, SNSAH, Jamia Hamdard
3 Assistant Professor, Department of Occupational Therapy, SNSAH, Jamia Hamdard
*Email Id: Lakshita182@gmail.com

Abstract -
Background: diaphragm dysfunction, known as ventilator induced diaphragmatic dysfunction, is a common but often overlooked complications of long term mechanical ventilation. it plays a major role in weaning failure; prolonged hospital stays in the intensive care unit and patient health complications.
Objective: this review explores the pathophysiology, clinical significance and physiotherapeutic interventions related to VIDD, emphasizing the integration of physical therapy into critical care management.
Methods: Databases including PubMed, Scopus, Cochrane Library, Google Scholar, BMC, NCBI, and Science Direct were searched using keywords such as "ventilator-induced diaphragmatic dysfunction," "physiotherapy," "inspiratory muscle training," and "early rehabilitation." Literature from 2010 to 2024 was reviewed.
Results: VIDD is mainly caused by oxidative stress, activation of proteolytic enzymes, and disuse atrophy. Physiotherapy approaches such as inspiratory muscle training (IMT), early mobilization, diaphragmatic breathing exercises, and ventilator optimization play a critical role in prevention and recovery. Diaphragm ultrasonography is a non-invasive method used for diagnosis and monitoring various conditions.
***Conclusion*:** Early physiotherapy intervention within ICU settings is pivotal in reducing the incidence and severity of VIDD. A multidisciplinary approach, incorporating respiratory therapists, physiotherapists, and intensivists, improves patient outcomes by promoting diaphragm function and facilitating successful weaning.

Keywords: VIDD, mechanical ventilation, respiratory physiotherapy, inspiratory muscle training, ICU rehabilitation, diaphragm atrophy

I. INTRODUCTION

VIDD is the term used to describe the diaphragmatic weakening and atrophy that directly result from controlled mechanical ventilation. This condition can cause prolonged stays in the intensive care unit (ICU), difficulties weaning, and impairments in respiratory muscle performance (1).
IDD describes the weakness and atrophy of the diaphragm that result from controlled mechanical ventilation, causing reduced function of respiratory muscles, challenges in weaning, and extended stays in the intensive care unit (ICU). (2,3).
The primary breathing muscle, the diaphragm, is highly active in normal physiological conditions. However, during mechanical ventilation, the contractile activity of the diaphragm is significantly reduced or even halted, especially in controlled environments. Within 18 to 72 hours of starting mechanical ventilation (MV), this lack of activity leads to rapid development of diaphragmatic atrophy. Research on both humans and animals' subjects has indicated that the diaphragm is more susceptible to atrophy from disuse compared to leg muscles. (4).
A frequently performed lifesaving procedure for critically ill patients suffering from respiratory failure, trauma, neurological damage, or major surgery is mechanical ventilation. Dependence on mechanical ventilation for an extended period has been associated with adverse effects with ventilator induced diaphragmatic dysfunction being the most significant, despite ensuring adequate gas exchange and reducing the effort needed for breathing.

Pathophysiology of VIDD:
VIDD develops through a number of interconnected mechanisms:

- Oxidative Stress: The diaphragm muscle fibers may produce more reactive oxygen species (ROS) as a result of mechanical breathing. Atrophy and damage to muscle fibers are caused in part by this oxidative stress (2).
- Proteolytic Pathway Activation: The diaphragm's contractile proteins degrade as a result of the activation of proteolytic systems, including as the calpain, caspase, and ubiquitin-proteasome pathways (3).
- Decreased Protein Synthesis: At the same time, protein synthesis pathways are downregulated, which exacerbates muscular atrophy (3).
- Altered autophagy and inflammation within diaphragmatic muscle fibres (3).

Prevalence of VIDD:
Table 1 highlights the prevalence of VIDD in different population.

Table 1: Prevalence of VIDD

Author	*Population*	*Ventilation duration*	*Prevalence of VIDD*
Levine et al., 2008(4)	Brain-dead organ donors	18–69 hours	50% diaphragm atrophy
Jung et al., 2014(5)	ICU patients	>48 hours	60–80% dysfunction

Zambon et al., 2016(6)	ICU post-surgical patients	≥24 hours	63% diaphragmatic dysfunction
Goligher et al., 2015(7)	ICU patients (weaning trial)	Variable	25–40% weaning failure due to VIDD

With major ramifications for respiratory therapy and early mobilization techniques, ventilator-induced diaphragmatic dysfunction (VIDD) has become a major problem in the fields of critical care and rehabilitation sciences. This thorough analysis looks at the pathophysiological causes of VIDD and how they relate to rehabilitation strategies meant to maintain diaphragmatic function and encourage functional recovery in patients on mechanical ventilation in a range of clinical settings. With an emphasis on evidence-based interventions to enhance diaphragmatic function and enable a smooth transition off of mechanical ventilation, the main goal of this study was to investigate the pathophysiology, clinical implications, and physiotherapy-based management strategies for ventilator-induced diaphragmatic dysfunction (VIDD).

II. METHODOLOGY

This review aligns with the primary objective of the study. Various academic databases such as PubMed, Scopus, web of science, Google scholar, Cochrane library, BMC and NCBI databases were utilized for an exhaustive search. the article was identified using a basic search strategy incorporating terms like "ventilator induced diaphragmatic dysfunction ", "VIDD", diaphragm dysfunction mechanical ventilation, mechanical ventilation diaphragm atrophy, and diaphragm muscle wasting ventilation. only articles published between 2010 and 2024 were included for this study.

Criteria for inclusion:

1. English-language literature only
2. about adult patients aged 18 years and older who are undergoing mechanical ventilation were considered.
3. Research focusing on the causes, diagnosis, clinical effects, and rehabilitation of VIDD
4. Is limited to studies involving human subjects only.
5. Therapies related to physiotherapy (such as IMT, diaphragm ultrasonography, and early mobilization),
6. Types of studies may encompass reviews, guidelines, observational studies, RCTs.

III. EVIDENCE SUPPORTING VIDD TO CLINICAL CONDITIONS

Pathophysiology for VIDD

1. *Critical Illness and Prolonged Mechanical Ventilation*

Rapid diaphragmatic atrophy and contractile failure can result from prolonged mechanical breathing, which is frequently required in critical illness. Increased oxidative stress and the activation of proteolytic pathways in the diaphragm muscle fibers are the causes of this dysfunction. These alterations impair the diaphragm's force production, which makes weaning off of ventilation difficult (8).

2. *Sepsis and Systemic Inflammation*

The diaphragm is especially prone to malfunction in septic patients because of systemic inflammation. In diaphragm muscles, the inflammatory response intensifies proteolysis and oxidative stress, resulting in structural damage and decreased contractility (9).

3. *Acute Respiratory Distress Syndrome (ARDS)*

ARDS patients frequently need mechanical ventilation, which can cause diaphragmatic weakening even if it can save their lives. Muscle atrophy and decreased endurance are caused by the combination of ventilator-induced diaphragm inactivity and lung damage (9).

4. *Neuromuscular Disorders*

VIDD is more likely to occur in people who already have neuromuscular disorders. In some patients, mechanical breathing can further weaken the diaphragm, making recovery more difficult and increasing the need for ventilatory assistance.

5. *Chronic Obstructive Pulmonary Disease (COPD)*

Because of their pre-existing respiratory muscle weakening, patients with COPD are especially vulnerable to VIDD. In these patients, mechanical breathing might hasten diaphragmatic atrophy, making acute exacerbation management more difficult and recovery durations longer.

IV. CLINICAL APPLICATIONS AND MANAGEMENT STRATEGIES FOR VIDD

1. *Lung and Diaphragm Protective Ventilation*

It is essential to use breathing techniques that safeguard diaphragm and lung function. Diaphragm damage can be lessened by modifying ventilator settings to avoid providing too much or too little support (10).

2. *Monitoring Diaphragm Function*

Early VIDD detection can be facilitated by routinely evaluating diaphragmatic function with instruments like ultrasonography. Parameters like diaphragm thickness and excursion can be monitored to provide useful information for prompt actions (11).

3. *Pharmacological Interventions*

There is potential in investigating pharmacological treatments that target the fundamental processes of VIDD, such as protease activity and oxidative stress. The effectiveness of substances like protease inhibitors and antioxidants in clinical settings is still being studied.

4. *Early Mobilization and Rehabilitation*

Diaphragmatic strength and function can be improved by incorporating respiratory muscle training and early mobilization into patient treatment programs. Reduced ICU stays and better weaning outcomes are linked to these strategies.

Goal setting for VIDD – enhanced overall functional independence

For patients with ventilator-induced diaphragm dysfunction (VIDD), establishing successful rehabilitation objectives is essential to improving total functional independence, easing ventilator weaning, and recovering respiratory function. When creating personalized and outcome-driven goals, doctors might

use the SMART framework—Specific, Measurable, Achievable, Relevant, and Time-bound (9).

Future Directions And Research Needs

Even though our knowledge of ventilator-induced diaphragm dysfunction (VIDD) is expanding, there are still a number of important areas that need more research. To improve treatment techniques and clinical strategies, further research is necessary. In particular, the following paths are advised:

1. ***Clarify Why the Diaphragm Deteriorates Faster Than Limb Muscles***

While all skeletal muscles experience disuse atrophy, the diaphragm atrophy during mechanical ventilation is noticeably more rapid and severe than that of limb muscles. This implies that the diaphragm is particularly vulnerable, which may be connected to its constant, high-frequency contractile action under healthy physiological settings. The goals of research should be: Examine the differences in the diaphragm's and leg muscles' molecular and cellular stress reactions during mechanical breathing.

Examine how respiratory and peripheral skeletal muscles differ in terms of oxidative metabolism, protease activation, and inflammatory signaling. Find out if this discrepancy is caused by the makeup of muscle fibres or innervation patterns. Designing protective measures tailored to individual muscles for patients in need of continuous mechanical breathing may be made easier with an understanding of these mechanisms (12).

2. ***Explore Mitochondrial Dysfunction in the Diaphragm***

One of the main characteristics of VIDD is the malfunctioning of mitochondria, which are essential regulators of muscle activity. According to studies, mechanical ventilation causes the following effects: Reduced electron transport chain (ETC) activity, increased mitochondrial reactive oxygen species (ROS) emission, and Damage to the structure and uncoupling of the mitochondria.

It is yet unknown, though, exactly how mechanical ventilation contributes to mitochondrial damage. Future studies ought to: Look into how autophagy, fusion/fission dynamics, and mitochondrial biogenesis relate to VIDD. Examine early indicators of ventilation-induced mitochondrial dysfunction.

Track real-time mitochondrial alterations in human and animal models using sophisticated imaging and metabolomic techniques.

These investigations will enable the development of mitochondria-targeted therapies and help determine whether mitochondrial dysfunction is a cause or a result of VIDD (13).

3. ***Identify Therapeutic Targets to Prevent or Mitigate VIDD***

There is an urgent need for therapeutic strategies that focus on the underlying molecular underpinnings of VIDD. Promising research directions include: Examining the effectiveness of antioxidants, especially those that target mitochondria, in reducing ROS-induced muscle injury; Investigating protease inhibitors, such as those that target calpain, caspase-3, and the ubiquitin-proteasome system, to prevent the degradation of important structural proteins like myosin and titin; and Since oxidative stress and proteolytic activation are central to diaphragm degradation. Assessing combination treatments to maintain diaphragm function, such as anti-inflammatory drugs, dietary assistance, and exercise-based techniques like Dynamic Neuromuscular Stabilization (DNS) (13).

V. DISCUSSION

Long-term use of mechanical ventilation can cause ventilator-induced diaphragmatic dysfunction (VIDD), a condition that impairs the diaphragm's structure and function. Disuse atrophy, oxidative stress, and the activation of proteolytic enzymes are the main causes of this dysfunction, which leads to the loss of diaphragmatic muscle fibers and decreased contractility. VIDD has significant clinical ramifications; it makes it more difficult to wean patients off of artificial ventilation, raises morbidity and lengthens ICU stays. Key tactics to prevent or reverse VIDD include early detection using diaphragmatic ultrasonography and the application of focused physiotherapy therapies, such as early mobilization and inspiratory muscle training (IMT).

By encouraging active diaphragmatic usage even when patients are still on ventilation, physiotherapists contribute significantly to the mitigation of VIDD. Weaning success can be improved and diaphragm function further protected by combining physiotherapy with ventilator methods (e.g., aided modes that engage spontaneous effort).

Future studies should focus on understanding mitochondrial contributions to dysfunction, identifying effective molecular-level treatment targets, and elucidating the diaphragm's unique sensitivity in comparison to other muscles. Translating these discoveries into clinical practice requires interdisciplinary cooperation between researchers, respiratory therapists, intensivists, and physiotherapists.

Although they need further research, emerging techniques including diaphragm pacing, phrenic nerve stimulation, and tailored pharmaceutical therapy show promise. A systematic and individualized strategy to regaining respiratory independence is also emphasized by using SMART goal-setting for rehabilitation.

Oxidative stress, proteolytic activation, reduced protein synthesis, and mitochondrial dysfunction are the main pathophysiological components of VIDD, underscoring the necessity of early detection and focused treatments. Patients with critical disease, sepsis, ARDS, neuromuscular abnormalities, and COPD are at increased risk.

Table 2: Strategies for the management of VIDD

Author name & year	Study title	Key findings
Zambon M., et al. (2016)	Assessment of Diaphragm Function and Weaning Outcome Using Diaphragm Ultrasound: A Review	Reviews the role of diaphragm ultrasound for assessing diaphragm function and its relationship with weaning outcomes from

		mechanical ventilation.
Martin AD., et al. (2011)	Inspiratory Muscle Strength Training Improves Weaning Outcome in Failure to Wean Patients: A Randomized Trial	Demonstrates that inspiratory muscle training (IMT) can improve diaphragm strength and reduce weaning time in ventilator-dependent patients.
Goligher EC., et al. (2015)	Diaphragm Dysfunction and the Impact of Early Mobilization on Ventilator Weaning Success	Early mobilization and rehabilitation help reduce VIDD severity and improve ventilator weaning success.
Bellani G., et al. (2014)	The Impact of Mechanical Ventilation on Diaphragmatic Function: A Review of Evidence	Review of literature on the pathophysiological effects of mechanical ventilation on diaphragm function and the need for preventive interventions.
Azoulay E., et al. (2017)	Diaphragm Weakness in ICU Patients: How to Predict and Manage It?	Focuses on predictive factors for diaphragmatic weakness and the role of physiotherapy in improving outcomes for mechanically ventilated patients.

Physiotherapy is essential in preventing and managing the conditions of VIDD:

1) ***Inspiratory muscle training:*** this training aimed at strengthening the diaphragm and improving the success rate of weaning outcomes(12). It includes:
   Protocol: typically involves 4-5 sets of 6-10 breaths through a threshold device, performed twice daily 5-7 days a week(12) .
   Intensity: training loads are often set at 30-40% of the patient's maximal inspiratory pressure(MIP).
   Outcomes : studies have shown improvements in MIP and tidal volume, however , effects on weaning duration and overall clinical outcomes are variable.
2) ***Early mobilization and rehabilitation****:* Early mobilization and rehabilitation, by following specific protocols, can help prevent diaphragm dysfunction. A well-organized, six-level program has been suggested: Level 0: passive turning every 2 hours for unconscious patients. Levels 1 and 2: joint exercises and assisted sitting for patients who are conscious. Level 3-5: progressive activities such as sitting on the edge of the bed, standing, and walking beside the bed for patients with tracheotomy. This method has been linked to enhanced diaphragm thickness and function (14).
3) ***Diaphragmatic stimulation techniques*****:** emerging interventions focus on direct stimulation of the diaphragm to preserve muscle function:
   Phrenic nerve stimulation: involves electrical activation of the phrenic nerve to induce diaphragmatic contractions.
   Diaphragm pacing : utilizes implanted electrodes to provide rhythmic stimulation, promoting muscle conditioning(15).
4) ***Ventilation strategies*****:** adjusting ventilator settings to encourage spontaneous breathing can help maintain diaphragmatic activity:
   Assisted modes: transitioning from controlled to assisted ventilation modes as early as feasible.
5) ***Pressure support ventilation*****:** employing lower pressure support levels to encourage patient-initiated breaths. A serious and little-known side effect of prolonged mechanical breathing is ventilator-induced diaphragmatic dysfunction (vied), which significantly worsens clinical outcomes such as longer intensive care unit admissions, delayed weaning, and elevated morbidity. The review highlights how the diaphragm's unique continuous workload and anatomical structure, which mechanical ventilation disrupts, make it highly susceptible to rapid atrophy and functional decline in comparison to peripheral muscles.

VI. FUTURE RESEARCH DIRECTIONS

- ***Why is the Diaphragm More Susceptible Than Limb Muscles*****?** Investigate metabolic and structural differences (12).
- ***Role of Mitochondrial Dysfunction:*** Real-time imaging and metabolomic studies are needed to explore ventilation-induced mitochondrial damage (13).
- ***Targeted Therapeutics*****:** Investigate protease inhibitors, mitochondrial antioxidants, and anti-inflammatory strategies to mitigate muscle wasting (13).

VII. CONCLUSION

Ventilator-Induced Diaphragmatic Dysfunction (VIDD) represents a significant barrier to successful weaning from mechanical ventilation and overall respiratory recovery. Insights gained from this examination underscore the importance of early identification and targeted rehabilitation strategies to mitigate diaphragmatic atrophy and weakness. Integrating evidence-based interventions—such as respiratory muscle training, early mobilization, and neuromuscular facilitation techniques—can preserve diaphragmatic function, reduce the duration of mechanical ventilation, and improve patient outcomes.

By emphasizing a multidisciplinary approach that includes physiotherapists, respiratory therapists, and occupational therapists, healthcare providers can enhance functional recovery, reduce ICU stay, and ultimately improve the quality of life in critically ill patients.

Finally, VIDD shouldn't be seen as a necessary side effect of mechanical ventilation. Instead, it is a condition that may be avoided and managed with prompt, evidence-based physiotherapy integration into intensive care unit care. Optimizing results and regaining the functional ability of critically sick patients will depend heavily on providing ICU teams with training, resources, and organized rehabilitation protocols.

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Lakshita Garg

- Completed Bachelors of physiotherapy from Manav Rachna International Institute of Research and Studies
- Pursuing Masters of physiotherapy from Jamia Hamdard